Diagnosis of Shock States

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KEYWORDS

• Shock • Echocardiography • Shock resuscitation phases • Mean systemic pressure
• Venous return • Preload responsiveness • Fluid accumulation syndrome
• Fluid removal tolerance

KEY POINTS

- Shock is a dynamic process with several phases requiring a specific approach during each phase. Echocardiography is an essential diagnostic tool in the management of shock.
- The initial goal is to diagnose the cause of shock, followed by treatment optimization, stabilization, and deescalation. Initial exam is rapid, qualitative, and based on pattern recognition. Subsequent examinations are often semiquantitative and quantitative. Non-echocardiographic modalities may offer additional insights. Preload responsiveness can guide fluid administration and removal. End-organ congestion can serve as a stopping point for fluid administration.
- Clinicians must be aware of limitations and common errors. Evidence for ultrasound improving patient outcomes is still evolving.

INTRODUCTION

Shock is a common[1] problem in the intensive care unit (ICU). It is defined as a life-threatening, generalized form of acute circulatory failure, associated with inadequate cell oxygen utilization and/or delivery, resulting in multiorgan failure and high mortality.[2]

Cell dysoxia develops when the oxygen supply cannot meet tissue demands, with oxygen consumption becoming dependent on the oxygen supply. Surrogate clinical markers of tissue hypoxia include skin mottling, capillary refill time, lactic acidosis, venous oxygen saturation, and venoarterial P_{CO_2} gradient. Blood pressure may or may not be decreased.[3]

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Abbreviations	
AR	aortic regurgitation
ARDS	acute respiratory distress syndrome
CO	cardiac output
EF	ejection fraction
ICU	intensive care unit
IVS	interventricular septum
LAP	left atrial pressure
LV	left ventricular
LVEDV	LV end-diastolic volume
LVESV	LV end-systolic volume
LVOT	left ventricular outflow tract
MAPSE	mitral annular plane systolic excursion
MI	myocardial infarction
MV	mitral valve
PE	pulmonary embolism
PLR	passive leg raising
Pms	mean systemic pressure
PmsR	Pms responsiveness
PR	preload responsiveness
Pra	RA pressure
RA	right atrium
RAP	right atrial pressure
RV	right ventricular
RVOT	right ventricular outflow tract
SV	stroke volume
SVR	systemic vascular resistance
TV	tricuspid valve
VR	venous return
VTI	velocity time integral

ECHOCARDIOGRAPHY IN SHOCK

Echocardiography is an indispensable tool for assessing patients with cardiopulmonary instability, providing direct information on anatomy and physiology.[4,5] Echocardiography is recommended over more invasive methods for the initial evaluation of shock.[2]

Compared with the traditional shock categories (hypovolemic, cardiogenic, obstructive, distributive),[1,2,6] echocardiography offers more granular diagnostic information with important implications. For example, echocardiography can easily differentiate between tamponade and pulmonary embolism (PE), both of which fall in the traditional category of obstructive shock. Because it offers additional information, echocardiography-guided shock management often differs from traditional management. For example, there has been a weak agreement between the prescription of fluid and inotropes/vasopressors between the traditional approach (Surviving Sepsis Campaign guidelines) and echo-guided resuscitation in sepsis.[7]

PHASES OF SHOCK, DIAGNOSIS, TREATMENTS, AND THE ROLE OF ECHOCARDIOGRAPHY

The echocardiographic goals during diagnosing and managing a patient with shock depend on the time point in the patient's illness. Four distinct fluid management phases exist in shock: salvage (resuscitation), optimization, stabilization, and

deescalation.[1,8] Echocardiography is typically used to determine shock mechanisms during the resuscitation phase and during the later phases shifts the focus more toward titration of therapies.[9]

Shock Mechanisms

Circulatory pressure can be measured directly during "no flow states" (cardiac arrest), or calculated in other ways, and is termed "mean systemic pressure" (Pms). When pumping, the heart redistributes this pressure so that it is high at its outlet (the aorta) and low at its inlet (right atrium [RA]), allowing the heart to fill due to the pressure gradient between the venules and the RA. Flow is opposed by any inlet impedance [commonly described by the equation VR = (Pms−Pra)/RVR]. When the heart is working efficiently, the circulation rate (cardiac output [CO]) is controlled solely by the Pms. The low-pressure right heart accepts as much venous return (VR) as the Pms allows and wants to give it until it is full in diastole. At this inflection point, Pra (RA pressure) increases, and the heart can fail. During failure, the heart cannot accept more VR, and the CO is determined by inotropy and chronotropy or how much afterload it needs to overcome. It is then intuitive that shock has 2 causes.

1. Cardiac failure
2. Low Pms

As Pms is determined by the volume and compliance of the vessels (particularly the splanchnic venules), low Pms itself has 2 causes—low volume within them or vasodilatation. *Both* result in reduced Pms and VR—a crucial fact that is often overlooked. If Pms is raised with fluid or vasopressors and stroke volume (SV) increases, the patient is said to be Pms responsive (the commonly used term "fluid responsiveness" is misleading as it only describes the volume component and not vessel compliance). Pms responsiveness (PmsR), also known as preload responsiveness (PR), is a normal physiologic state. It does not mean that increasing Pms is indicated. Mean arterial pressure and CO also have to be taken into account.

The main causes of heart failure and low Pms are listed in **Table 1**. The core approach to shock diagnosis and management, based on these 2 categories, is shown in **Fig. 1**.

Fluid, Vasopressor, and Inotropic Management Strategy During Different Phases of Shock

Familiarity with time-varying fluid management strategy, early on in shock is essential. Patients are often initially seen by ICU clinicians throughout various phases of shock and will need a tailored therapeutic approach to therapy. During the resuscitation phase, the patient may be in a life-threatening condition with the focus being on patient rescue. Here, fluids may be given more liberally in the form of boluses. During the optimization phase, the patient is stabilizing but still at risk of deterioration. The focus at this stage is on organ rescue by optimizing CO to ensure oxygen delivery and end-organ perfusion, while minimizing fluid creep. Fluids are given more cautiously here, guided by the Pms (preload) responsiveness of the patient. During the stabilization phase, the patient has emerged from the shock state, and the emphasis is on maintaining organ homeostasis. Here, fluids should only be given as maintenance to replace losses. In the deescalation phase, the focus is on end-organ recovery. Fluids may be removed if there is evidence of end-organ congestion. Differentiation exists between the deescalation (fluids are stopped and or passively removed) and deresuscitation phases (fluids are actively removed). There are some opinions that the term "deresuscitation" should be removed.[10,11] Regardless of the

Table 1
Causes of shock as they relate to heart failure versus low Pms

Heart Failure	Low Pms
Extremes of heart rate; arrhythmias	Vasodilatation
Poor contractility	• Infection
Acute cor pulmonale	• Systemic inflammatory response
Valve dysfunction (including dynamic LVOT obstruction)	• Major surgery
	• Anaphylaxis
Tamponade/constrictive pericarditis	• Loss of sympathetic tone (anesthesia, sedation, spinal cord injury)
Aortic dissection	Hypovolemia
	• Bleeding
	• Gastrointestinal losses
	• Anaphylaxis (capillary leak)
	• Renal replacement therapy (fluid removal exceeding intravascular refill rate)
	• Systemic capillary leak syndrome (Clarkson disease)

terminology, 2 principles apply. First, fluids should be removed if there is evidence of excessive fluid accumulation, either passively or actively using diuretics/ultrafiltration. Second, as fluids are actively removed, the patient must be monitored for tolerance, since the risk of aggressive fluid removal is transition into a state of lower Pms and resultant hypoperfusion. Vasopressors are often used in parallel with intravenous fluid in the salvage phase (to recruit unstressed volume into stressed volume, thus increasing effective preload). Inotropes are usually initiated after diagnosing cardiac dysfunction associated with the hypoperfusion during the optimization phase (Table 2).

Echocardiography Goals During Different Phases of Resuscitation

During the salvage (resuscitation) phase, the echocardiography examination is based on 2-dimensional (2D) views and focuses on recognizing shock patterns (phenotypes). It can guide initial fluid resuscitation and other more specific critical treatment decisions. Certain Doppler techniques (left ventricular outflow tract [LVOT] velocity time integral [VTI] to estimate SV and color Doppler to screen for major valve regurgitations) can also be used early on. During the optimization phase, echocardiography adds perfusion measurements (SV), PR, and many other quantitative parameters to guide the titration of fluids, inotropes, and vasopressors. During the stabilization and deescalation phases, the focus shifts from optimizing perfusion to identifying and preventing complications (measuring lung and venous congestion) and avoiding hypoperfusion during fluid removal (identifying patients who would not tolerate it) (see Table 2; Table 3). Details of ultrasound techniques relevant to shock will be discussed next.

Role of Non-echocardiographic Ultrasound Modalities

Nonechocardiographic ultrasound findings are sometimes, but not always, useful in shock management. The indication for nonechocardiographic ultrasound modalities depends on the shocked patient's circumstances. The lung ultrasound finding of B-lines is discussed in detail in the following section. Ultrasound findings suggestive (absent sliding, absent lung pulse, absent vertical artifacts) or diagnostic (present lung point) of pneumothorax[12,13] in an unstable patient are useful. A time-consuming examination should not delay urgent chest decompression if the diagnosis is already obvious or suspected based on other findings. Finding a deep vein

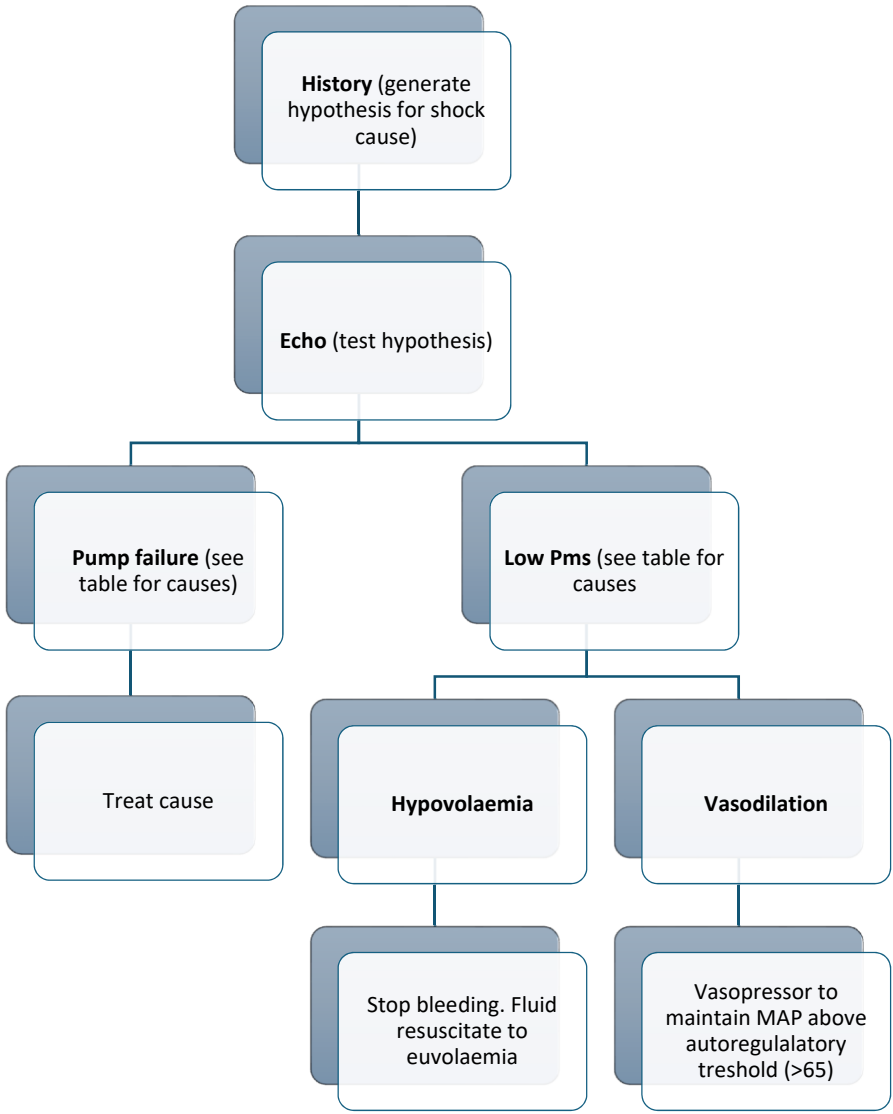
History (generate hypothesis for shock cause)
Echo (test hypothesis)
Pump failure (see table for causes)
Low Pms (see table for causes
Treat cause
Hypovolaemia
Vasodilation
Stop bleeding. Fluid resuscitate to euvolaemia
Vasopressor to maintain MAP above autoregulalatory treshold (>65)

Fig. 1. Basic diagnostic and therapeutic approach to shock.

thrombosis on venous ultrasound[14] in a shocked patient with acute or pulmonale identified on echo is highly suggestive of PE. A negative FAST examination in a trauma shock scenario excludes intraabdominal bleeding as the cause of the patient's condition.[15]

SALVAGE (RESUSCITATION) PHASE

Identification of Shock Patterns

The initial goal is to rapidly diagnose the cause of shock by identifying critical echo patterns (tamponade, hypovolemia, left ventricular [LV] and right ventricular [RV] failure, catastrophic valve dysfunction, and indirectly, decreased vasomotor tone). The

Table 2
Ultrasound techniques and goals of fluid management during shock phases

Shock Phase	Resuscitation (Salvage)	Optimization	Stabilization	Deescalation
Shock characterization	Life-threatening shock	Compensated shock High risk for decompensation	Shock absent Vasopressor at low stable rate may still be needed	Shock absent Vasopressor at low stable rate may still be needed
Goal	Patient rescue	Optimize oxygen delivery	Maintain organ homeostasis	Organ recovery
Ultrasound technique	2D (IVC, LV size)	2D (IVC, LV size) PR (VTI variability, VTI PLR)	2D (IVC, LV size) Lung congestion, EVLW (B-pattern) Venous congestion (VEXUS)	2D (IVC, LV size) PR (VTI variability, VTI PLR) Lung congestion, EVLW (B-pattern) Venous congestion (VEXUS)
Fluid strategy	Liberal fluids (fluid bolus) if hypovolemia suspected	Optimize fluids (fluid challenge), stop bolus Avoid fluid creep	Start fluid removal Start negative fluid balance goal	Active fluid removal Set negative fluid balance goal
Ultrasound is used for decision on	When to start fluid	When to stop fluid	When to start removing fluid	When to stop removing fluid

The fluid management strategy during different phases of shock is a dynamic process with changing priorities over time. Ultrasound can be used for decision-making. During the salvage phase, ultrasound is used for decisions to give fluids. During the optimization phase, fluids are stopped and given only based on ultrasound tests of preload responsiveness. During the stabilization and deescalation phases, fluids start to be removed, initially passively or, if needed, actively using diuretics or ultrafiltration. Ultrasound is used to guide fluid removal by identifying end organ congestion and identifying those patients who might not tolerate fluid removal.

priority focuses on assimilation of signs to indicate potential causation of the shock state using basic 2D views, rather than time-consuming quantitative measurements. The clinician should start with a pretest differential diagnostic list of mechanisms and causes of shock, based on available clinical information, before doing the echo examination. Adapting the approach to pretest probability[16,17] based on the specific clinical setting with specific common causes of shock is often helpful. For example, in emergency department (ED), sepsis is the most common cause of undifferentiated shock, whereas tamponade is rare.[18] Similarly, in the general ICU population, sepsis is the most common, followed by hypovolemia (mostly from bleeding) and cardiogenic shock (mostly from myocardial infarction and cardiomyopathy).[19] The relative distribution of various shock types will differ in specialized ICUs (eg, cardiothoracic surgery, neuro ICU).

The sequence of patterns is important since certain critical findings, although uncommon, should prompt immediate treatments and/or consults. For this reason, the

Table 3
Basic echo patterns in shock, their differential diagnosis, and treatments

Echo Pattern	Qualitative Findings (Basic)	Additional Findings	Differential Diagnosis	Therapeutic Considerations
Tamponade	**Pericardial effusion** (circumferential) **Cardiac chamber collapse**	**Systolic collapse of RA (>1/3 cardiac cycle)** **Diastolic collapse of RV** **LV or LA collapse** **Dilated IVC** **Large hemothorax/pleural effusion** **Mediastinal mass with compression of PA**	Pericardial circumferential effusion (bleeding, uremia, malignancy, autoimmune) Regional compression (hematoma s/p cardiothoracic surgery; large hemothorax/pleural effusion; solid masses)	Pericardiocentesis/pericardial window Resternotomy after pericardiotomy Evacuation of hemothorax, pleural effusion
Tension pneumothorax	**Absent: lung sliding, lung pulse, B-lines** **Dilated IVC**	**Variably present if tension: lung point**	Tension pneumothorax (positive pressure ventilation) Other causes of absent lung sliding	Chest tube/thoracentesis
Catastrophic valve failure *Severe MV or AV regurgitation or stenosis*	**Grossly abnormal MV or AV appearance** **Severe regurgitation (CFD)**	**Valve destruction** **Ruptured papillary muscle** **Flail leaflet** **Gross coaptation defect** **Severe valve thickening and restriction**	Mitral regurgitation: endocarditis, myocardial ischemia, trauma, myxomatous disease Aortic regurgitation: endocarditis, aortic dissection, myocardial ischemia Aortic stenosis	Cardiothoracic surgery consultation Mechanical circulatory support (no IABP if AR)
LVOT obstruction	**Systolic anterior motion of mitral anterior leaflet**	**Hyperdynamic LV EF**	LVOT obstruction (hypovolemia, LV hypertrophy, sigmoid septum in elderly, hypertrophic cardiomyopathy, catecholamines) Post-CT surgery	IV fluid, alpha mimetic, avoid inotrope

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Table 3
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Echo Pattern	Qualitative Findings (Basic)	Additional Findings	Differential Diagnosis	Therapeutic Considerations
Aortic dissection	**Ascending aortic flap**	**Pericardial effusion** **Aortic regurgitation**	Aortic dissection	Cardiothoracic surgery consultation
Hypovolemia	**Hyperdynamic small LV** **Kissing papillary muscles** **Small fully collapsible IVC** (spontaneously breathing) **Small significantly collapsible IVC** (mechanically ventilated)	**LVOT VTI or AoVmax variability** (mechanical ventilation) **E/A<0.8, E/e'<8** **Positive FAST**	Bleeding Sepsis (early phase) Gi fluid losses	Crystalloid Colloid Blood product
LV failure	**LV systolic dysfunction**: visual estimation of LV EF (normal vs mild/ moderate, vs severely reduced<40%)	**LV dilatation** **LV hypertrophy** **LA dilatation** **Shift of interatrial septum to right** **Regional wall motion abnormalities** (in coronary artery territory; apical ballooning) **E/A>2, E/e'>14**	Global dysfunction (dilated cardiomyopathy; myocarditis; sepsis) Regional dysfunction (myocardial ischemia; takotsubo cardiomyopathy)	Inotrope Revascularization Mechanical circulatory support

RV failure	**RV dilatation**	**Tricuspid regurgitation** (indicating increased PASP) **RV dilatation with septal flattening and dyskinesia** **RV systolic dysfunction:** visual estimation of TV annulus motion +/− **LV inferior wall hypokinesis** (RV infarct) **Shift of interatrial septum to left** **TAPSE** <17	Acute cor pulmonale (pulmonary embolism; ARDS) Acute on chronic cor pulmonale RV infarction Biventricular failure	Inotrope Vasopressor Diuresis/renal replacement therapy Adjust mechanical ventilation setting (PEEP, tidal volume) Anticoagulation/ thrombolysis/ embolectomy Pulmonary vasodilator ECMO
Decreased vasomotor tone	**Normal or hyperdynamic LV EF** **LV end-diastolic area not at extremes** (LVEDA >5.5 cm^2, absence of kissing papillary muscles) **IVC not at extremes** (IVC is not very small and not completely collapsible) -spontaneously breathing	**No LVOT VTI or AoVmax variability** (mechanical ventilation) **E/A>1**	Sepsis Adrenal insufficiency End-stage liver disease Vasodilators	Vasopressor Stress dose hydrocortisone Source control in sepsis Antidote

Bold: essential information; Not bold: further expansion of information.

authors recommend ruling out obstructive shock (eg, tamponade, tension pneumothorax) and catastrophic valve problems (eg, ruptured papillary muscles with severe mitral regurgitation) first (Fig. 2). At the same time, the entire basic echocardiographic examination should not take more than a few minutes, during which all the patterns should become obvious. Simultaneous efficient image acquisition, interpretation, consideration of differential diagnosis, and planning of critical treatment decisions are all done at the bedside and by ICU clinicians. This is an integral and unique part of point-of-care ultrasound. Specific findings with their differential diagnoses and treatments are shown in Table 3.

The second important goal during the salvage phase is the avoidance of any iatrogenic complications. Examples include delays in timely revascularization of patients with regional wall motion abnormalities, delaying diuretic and inotrope therapy in patients with dynamic LVOT obstruction, and tardiness in stopping unnecessary fluid administration in overt decompensated left or right heart failure.

Technical Aspects

Initial echo examination depends heavily on basic 2D imaging; it is goal- and problem-oriented, limited in scope, and qualitative or semiquantitative. At the same time, it should be systematic. Several basic echocardiographic views are preferred (PLAX, PSAX, A4C, SC).[20] One view (eg, subcostal) may be adequate in an emergency (eg, cardiac arrest).[21,22] The subcostal view can yield richer information from a single view, since it shows both cardiac structures and the inferior vena cava (IVC). Coupled with lung views, it can be used for more granular shock classification[23] (Fig. 3). Detailed descriptions of views in general and specifically in ICU are without the scope of this work and can be found elsewhere.[20,22] Alternative views can be obtained in situations where standard views are unobtainable.[24] Recognition of echo patterns in shock has been an integral part of critical care echocardiography since the early days of ICU ultrasound.[25] There have been iterations within many shock algorithms and protocols.[23,26–30] These are of varying complexity but commonly use combinations of cardiac, lung, venous, and abdominal/other views, with or without

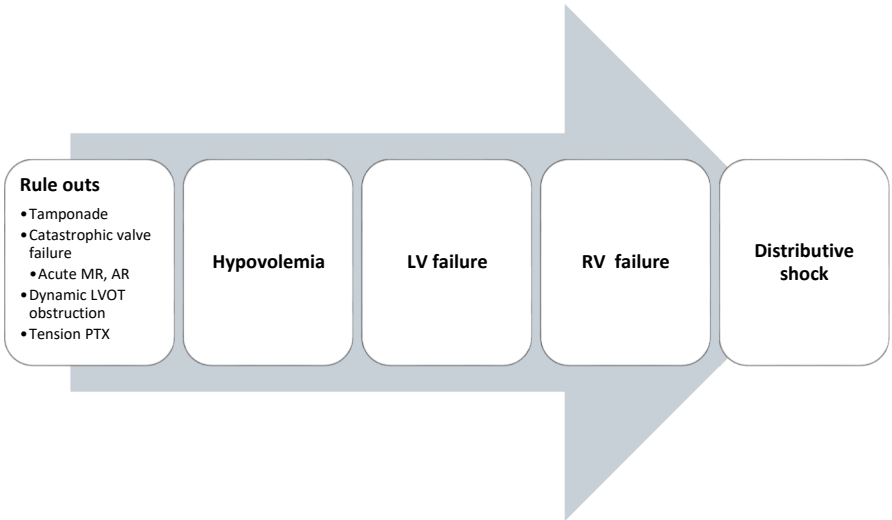

Fig. 2. Sequence of echocardiographic patterns during salvage phase of shock. See text for details.

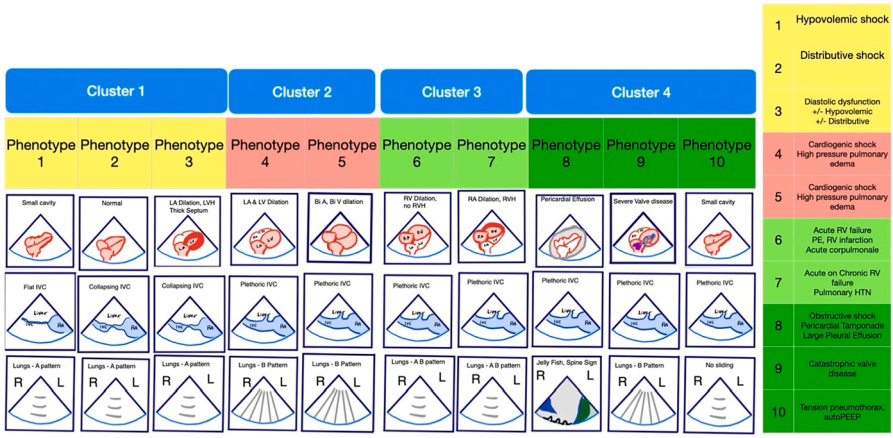

Fig. 3. EASy phenotypes. EASy phenotypes are based on the subcostal 4-chamber view (*top row*), subcostal IVC view (*middle row*), and lung evaluation in the upper lung fields (*bottom row*). Phenotypes 1 to 3 are grouped within cluster 1; phenotypes 4 and 5 are part of cluster 2; phenotypes 6 and 7 are part of cluster 3; and phenotypes 8 to 10 part of cluster 4. In general, cardiac assessment assesses myocardial performance, IVC evaluation provides the estimate of preload responsiveness, and lung examination defines fluid tolerance (A-profile) and intolerance (B-profile). Phenotype 1 describes the patient with hypovolemic shock with small diastolic ventricular chamber size, collapsed IVC with significant variability in its diameter throughout the respiratory cycle, and A-profile on lung examination. Phenotype 2 is consistent with distributive shock in adequately fluid-resuscitated patient with good cardiac filling, normal IVC size, and A-profile on lung examination. Phenotype 3 describes patient with hypertrophic LV, commonly dilated left atrium, small and collapsible IVC, and A-profile on lung examination. Phenotypes 4 and 5 describe patient with isolated LV (phenotype 4) and biventricular (phenotype 5) dysfunction, usually plethoric IVC and B-profile on lung examination. Phenotype 6 most commonly describes patient with ARDS complicated by RV dysfunction. These patients demonstrate enlarged RV, plethoric IVC, and B-profile on lung examination more commonly reflecting nonhydrostatic edema. Phenotype 7 includes patients with underlying pulmonary hypertension and RV hypertrophy who develop acute or chronic RV failure. These patients have enlarged and hypertrophic RV, plethoric IVC, and variable lung profile (B-profile shown here). Phenotype 8 shows pericardial tamponade, phenotype 9 shows severe valvular regurgitation, and phenotype 10 shows tension pneumothorax with loss of lung sliding and small LV cavity. Bi A, Bi atrial; Bi V, Bi ventricular; HTN, hypertension; LA, left atrium; LVH, left ventricular hypertrophy; RA, right atrium; RVH, right ventricular hypertrophy. (*Courtesy of* N. Bughrara, MD, Albany, NY).

quantitation. An observational study identified 5 cardiovascular clusters in sepsis (well resuscitated, hyperkinetic, hypovolemic, LV dysfunction, RV dysfunction), mirroring the abovementioned basic shock patterns.[31]

Estimating Cardiac Output During the Salvage Phase

SV and CO can quantitatively inform forward cardiac flow. SV is the difference between LV end-diastolic volume (LVEDV) and LV end-systolic volume (LVESV). LVEDV is determined by preload, compliance, ventricular remodeling, and LV-RV interaction. LVESV is determined by afterload, remodeling, and contractility.[32] Therefore, quantifying the SV will enable the clinician to recognize low SV and CO states and identify any modifiable factors (with emphasis on preload, afterload, and contractility) responsible for SV derangement. LVOT VTI is the echocardiographic surrogate for SV and

CO. Because flow is such a crucial indicator and the target of most treatments, LVOT VTI measurement should be used as part of a basic echo examination. Indeed, it has been proposed that every operator at every stage of training learns it.[33]

Most adults have an LVOT diameter of 2 cm.[34] It is assumed that LVOT area is stable, and changes in SV are mostly a result of changes in LVOT VTI. This is important for dynamic tests and will be discussed in the following stages of resuscitation. Static VTI values are also useful, even in the initial phase, because they allow the categorization of patients into either low or normal/high SV groups. Details of the examination technique have been described elsewhere. Briefly, A5C or A3C views are used, LVOT is interrogated with color Doppler interrogation (because the VTI technique cannot be done in moderate to severe aortic regurgitation (AR) or LVOT obstruction with high flows), and pulsed wave doppler is placed at the base of the aortic valve leaflets, while keeping the beam as parallel as possible with LVOT. Several high and low VTI cutoffs have been proposed in critical care medicine literature. One reasonable approach is assuming that VTI less than 14 defines low SV (because SV is <60 even with LVOT diameter up to 2.3 cm) and VTI greater than 22 defines normal/high SV (because SV is >60 even with LVOT diameter down to 1.9 cm).[35] Another approach proposed 16 to 20 cutoffs. In most recent cardiology literature, the range of normal values is even wider (13.3–27.3 for men, 13.6–29.6 for women).[36] If the LVOT is unavailable, it is possible to use right ventricular outflow tract (RVOT) VTI using the same logic but with a slightly different cutoff value (15 cm for the lower limit of RVOT VTI) since the RVOT diameter is larger than the LVOT diameter.[37] To quantitate SV, assuming LVOT diameter 2 cm, SV can be easily calculated as LVOT VTI x 3.14. **Fig. 4** shows the role of LVOT VTI in the initial phase of shock resuscitation.

There are several specific diagnostic and therapeutic reasons why measurement of SV (VTI) in the early salvage phase of shock might be essential beyond just complementing the information obtained from 2D patterns. For diagnosis, first let us consider the scenario of normotensive shock (hypoperfusion demonstrated by, eg, lactic acidosis), a serious and transient state that often deteriorates into a full hypotensive shock state (mean arterial pressure deterioration after compensatory mechanisms are overwhelmed). Low VTI was a sensitive and powerful predictor of mortality in normotensive shock patients on cardiac ICU.[38] As a sole parameter, VTI assisted in identifying high-risk patients. Second, let us consider the scenario of distributive shock (specifically sepsis). Here, we see an inappropriately normal ScVo2 from impaired tissue O2 uptake, despite cardiac dysfunction. This may divert the examiner away from the true diagnosis of decreased CO (a low VTI will point toward the correct diagnosis). Third, several situations may be encountered in the LV failure/low ejection fraction (EF) scenario. One is that of cardiomyopathy with chronically, severely reduced LVEF and superimposed sepsis. In this not-so-uncommon situation, a normal or even high SV (VTI) generated by severely enlarged LV (with reduced EF)[32] supports the diagnosis of distributive shock; LV dysfunction is just a bystander. Another one is of LV systolic dysfunction with an acute LV insult (eg, myocarditis or severe diastolic dysfunction) with just a mild LVEF and severely reduced SV. LVEF is highly variable in cardiogenic shock,[39] not informative on ventricular performance or hemodynamics.[40] At the same time, SV (VTI) circumvents shortcomings of LVEF and will inform on the true degree of low-flow state. Fourth, in a scenario of hyperdynamic LV, normal or high SV (VTI) suggests the patient is fluid-replete and should not be treated as hypovolemia. For treatment decisions, identification of low VTI, paired with structural cardiac dysfunction and tissue hypoperfusion, should trigger the decision to start inotropes. In contrast, normal VTI, despite structural cardiac dysfunction, does not require intervention with inotropic or mechanical devices. The not-so-uncommon

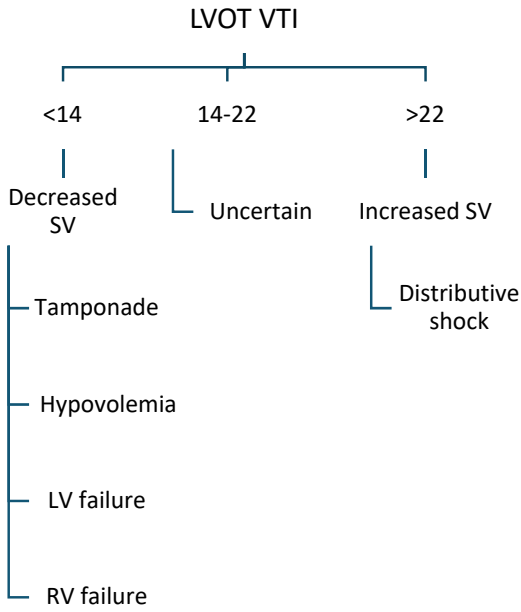

Fig. 4. Role of LVOT VTI during salvage phase of shock. Use of left ventricular outflow tract (LVOT) velocity time integral (VTI) as an initial step in diagnosing shock mechanism. LVOT VTI should be accompanied by a basic 2D examination. Decreased stroke volume (SV) can have multiple different causes. If the LVOT VTI is 14 to 22, diagnosis is less certain. For further discussion on LVOT VTI variability (not shown), see text and Fig. 3.

"gray" zone of LVOT VTI 16 to 20[37] (or 14–22),[35] paired with hypoperfusion, is where the most challenging situations in diagnosis and management will occur. These require careful consideration of interventions to optimize SV and O2 content[37] to avoid myocardial stress/oxygen consumption and arrhythmia from overzealous inotropic support. Lastly, the variability of LVOT VTI can indicate fluid tolerance (or other problem) (Fig. 5). This will be discussed in the mean systemic pressure (or preload) responsiveness section.

Additional Findings During the Salvage Phase

Echocardiographic findings are not dichotomous but represent a continuum of additional 2D and Doppler findings. As the echocardiographer's skills increase, additional techniques and findings (see Table 3) can be easily incorporated into the early phase. Specifically, noticing the 2D examination signs of chronicity (chamber dilatation and hypertrophy), measurement of RV function (TAPSE), measurement of LV diastolic function (E and A mitral inflow velocities and e' tissue velocity of mitral valve [MV] annulus), and LVOT VTI variability as an indicator of Pms responsiveness can be done early on, without significantly delaying the initial examination in the hands of more experienced ICU clinicians.

OPTIMIZATION PHASE

The main goal at this point shifts from rescuing the patient to optimizing organ perfusion. Echocardiography during this phase is used in 2 ways. First, it is used to assist titration of vasopressors, inotropes, and fluids. Second, to track the changes in the

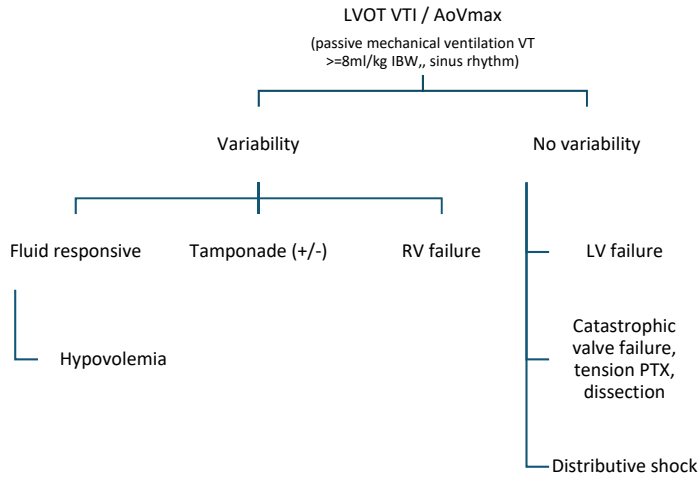

Fig. 5. LVOT VTI/AoVmax variability in preload responsiveness states and its mimics. LVOT VTI (or aortic flow peak velocity AoVmax) variability can indicate preload responsiveness but also other problems. RV failure may result in LVOT VTI variability from cyclic afterloading of RV while on mechanical ventilation. In tamponade, LVOT VTI variability occurs because of ventricular interdependence and may be lost on mechanical ventilation.

disease state (either tracking the course of the original problem or diagnosing a new problem). Any unexpected hemodynamic change should prompt reexamination. Septic shock is a dynamic process with several possible mechanisms[31] that can change and/or overlap over time. Third, echo is used to evaluate hemodynamic responses to treatment. Examples are demonstrating a change of LVEF secondary to a vasopressor-induced increase in LV afterload, RV dysfunction secondary to positive pressure ventilation, and acute respiratory distress syndrome (ARDS)-induced increase in RV afterload. This phase allows more time to obtain additional data, using advanced techniques to refine the diagnosis (**Table 4**). The specific nuances and limitations of individual parameters especially relevant to expert shock management will be discussed next.

Tamponade

When a pericardial effusion exerts enough pressure, the lowest pressure chambers (RA then RV) will be compressed, impairing their filling and output—a state known as cardiac tamponade. Pericardial effusions are easy to identify with ultrasound. Although tamponade is a clinical diagnosis, there are several sonographic features. Right-sided chamber collapse, significant velocity variation through the mitral or tricuspid valves (TVs), signs of venous congestion, and a heart swinging within an effusion are all highly suggestive. Here, the use of vasopressors or volume to increase preload is indicated as a bridge until urgent ultrasound-guided drainage of the effusion is undertaken.

There are important caveats. The variability of MV and TV inflow seen in spontaneously breathing patients will not apply to mechanically ventilated patients. The dyskinetic motion of interventricular septum (IVS) and relaxation impairment have been suggested instead.[41] The presence of pulmonary hypertension may prevent RV and RA collapse. In addition to pericardial effusion, localized compression from hematoma, mediastinal mass, hemothorax, and pleural effusion, including empyema, can all result in tamponade. Transesophageal echocardiogram must be obtained in

Table 4
Semiquantitative and quantitative techniques in shock

Shock Subgroup	Semiquantitative and Quantitative Techniques
Tamponade	***TTE*:** **Size of effusion** (does not equal its hemodynamic effect); **speed of development is important** **Variability of tricuspid valve and mitral valve inflow** (variability of inflow more than 30% [MV], 60% [TV]) (PW) **Pleural effusion, solid mass** (causing cardiac chamber compression) ***TEE after pericardiotomy:*** **Pericardial effusion (localized)/hematoma with cardiac chamber compression**
Valve failure (general)	**Primary vs. functional regurgitation; chronic vs. acute** **Infective endocarditis** (vegetation, abscess, valve perforation) **Grossly abnormal prosthetic valve dysfunction** (large central regurgitation, periprosthetic regurgitation, prosthetic instability)
Severe mitral regurgitation	**Secondary LV changes**: normal/hyperdynamic LV EF and LV size (acute MR) vs dilated LA/LV (chronic MR) **Large central jet** (>50% LA) or **eccentric wall impinging jet of variable size** (CFD) **Large** (>1 cm) **flow convergence MR throughout systole** (CFD) **Vena contracta width** ($\geq$7 mm) (CFD) **Holosystolic/dense triangular jet** (CW) **PV systolic flow reversal** (PW) **MV inflow: E-dominant** (PW) *Caveat:* in acute MR, LV is not dilated; jet often eccentric; rapid equalization of pressures >low regurgitant volume; short duration of jet; low velocity may underestimate severity
Severe aortic regurgitation	**Secondary LV changes**: normal LV size (acute AR) vs dilated LV (chronic AR) **Large jet width** (color Doppler); **AR jet width** (PLAX) (jet width/LVOT width >65%) (CFD); **AR jet area** (PSAX) (jet area/LVOT area >60%) (CFD) **Large flow convergence** (CFD) **Vena contracta width** (>6 mm) (CFD) **Dense jet** (CW) **Steep deceleration** (pressure half time < 200 msec) (CW) **Prominent descending aorta holodiastolic flow reversal** (PW) *Caveat*: in acute AR, LV will not be dilated; color jet may not be impressive; vena contracta is better; clinical suspicion!
Severe aortic stenosis	**AV jet peak velocity** (Vmax >4 m/s) (CW) **AV mean gradient** (>40 mm Hg) (CW) **AV area** (<1.0 cm^2, indexed <0.6 cm^2/m^2) (PW/CW) **Dimensionless index (velocity or VTI ratio) (LVOT Vmax/AV Vmax or LVOT VTI/AV VTI)** <0.25 (PW/CW) *Caveat*: special discordance situations (low-flow (EF<50%, SVI<35) low gradient (Vmax<4, MG<40) with AVA<>1.0; high-flow (SVI>58) high gradient (Vmax>4, MG>40) with AVA>1.0
LVOT obstruction	**Turbulent flow** (CFD) **Associated mitral regurgitation** (CFD) **Late-peaking velocity curve**: concave upward in early systole ("dagger"-shaped appearance) of LVOT outflow (CW)

(*continued on next page*)

Table 4 (*continued*)

Shock Subgroup	Semiquantitative and Quantitative Techniques
Aortic dissection	**Segmental wall motion abnormalities** **Aortic regurgitation** (CFD) ***TEE:*** **Ascending aortic flap**
Tension pneumothorax	*Caveat*: lung point may not be present if large tension pneumothorax
Hypovolemia	**Preload (Pms) responsiveness:** IVC techniques: **Distensibility of IVC** (ΔIVC $\geq$ 12%–18%)[a] **Collapsibility of IVC** (ΔIVC $\geq$ 48%) **Collapsibility of SVC** (ΔSVC $\geq$ 36%)[a] LVOT VTI techniques: **Variability of aortic flow velocity** (ΔAoVmax $\geq$ 12% or ΔLVOT VTI $\geq$ 18%)[a] **Increase of LVOT VTI** (passive leg raise or fluid bolus) (ΔLVOT VTI $\geq$ 12.5%) **Mini-bolus** (ΔLVOT VTI $\geq$ 15%) **End-expiratory to end-inspiratory occlusion change of LVOT VTI** (ΔLVOT VTI $\geq$ 13%)[a]
LV failure	**LVOT VTI** (PW) (14–22 cm) **LV dimensions (signs of chronicity)** (thin LV walls; dilatation of LV (LVIDd > 6 cm); LV hypertrophy (IW: >1.0 cm, IVS: >1.1 cm) (2D or M-mode) **LV systolic function** **Global:** visual estimation of LV EF (<30%, 30%–40%, 40%–50%, 50%–70%, >70%)/Simpson **Regional: RWMA (regional wall motion abnormalities)** **MAPSE** (M-mode): <10 (decreased) **S'** (tissue Doppler): <7.5 (EF<50%) vs >7.5 (EF>50%) **LV diastolic function** **Mitral E/A** (PW) (E/A<0.8 = relaxation impaired/LVEDP normal vs E/A>2.0 = restrictive, LVEDP high) **Mitral annulus E/e'** (tissue Doppler): E/e' <8 (normal LVEDP) vs >13 (increased LVEDP)
RV failure	**RV dimensions (dilatation)** (RV base diameter >4.1 cm (A4C), RVOT diameter >3.5 (PLAX, PSAX) **RVEDA/LVEDA ratio** (in A4C) (dilatation: >0.6, severe: >1.0) **Interventricular septal flattening, septal dyskinesia** (PSAX) **RV function: TAPSE** (M-mode): <17 (decreased), **S'**(tissue Doppler): <17 (decreased), **FAC** (fractional area change): <35% (decreased) **Sign of chronicity: RV hypertrophy** (>5 mm) (SC4C), **aneurysms** **Pulmonary artery pressures** (PASP—from TR, PADP—from PR, PAMP—various methods; midsystolic notching) Associated findings: **LV systolic dysfunction, LV diastolic dysfunction, left-sided valvular abnormalities** **Displaced septal position of tricuspid valve** **Clot in transit; regional wall motion: McConnell sign** (RV hypokinesis with apical sparing); **60/60 sign** (PASP<60 mm Hg/ RVOT AT<60 msec) (CW/PW) **Shunt detection**: bubble study (PFO), ultrasound enhancing agents (VSD)
Decreased vasomotor tone	**Absence of preload responsiveness** suggests distributive shock without hypovolemia

[a] Requires passive mechanical ventilation.
Bold: essential information; Not bold: further expansion of information.

postcardiothoracic surgery patients with suspected tamponade if transthoracic echo does not convincingly rule it out. "Low-pressure" tamponade may respond to volume. Intubating a patient with tamponade may be potentially dangerous as RV filling will be further restricted from the effect of positive pressure and increased intrathoracic pressure (see **Table 4**).

Mean Systemic Pressure Responsiveness

Ordinarily, Pms controls VR with an efficient heart, keeping right atrial pressure (RAP) low, thus maintaining a pressure gradient for VR. Pms is determined by vascular compliance and volume. The primary site of this control is the splanchnic venules. These venules contain a significant proportion of the circulatory volume and are highly innervated with alpha receptors, meaning the sympathetic nervous system can easily control their compliance and, therefore, pressure. Low Pms causes reduced VR, resulting in low CO. It, therefore, stands to reason that both hypovolemic and vasodilatory shock (before increasing Pms with resuscitation) have identical appearances with ultrasound—a poorly filled (low volume), hyperdynamic heart. In a circulatory system with a nonsucking passively filling pump (like the heart), resistance has a negligible effect on pump output if it is located near the pump with a poorly compliant bed upstream (ie, at arterial level)—this is why hypertension or even aortic cross-clamping has only a small effect on CO. In contrast, if resistance is closer to the pump inlet with high compliance upstream (such as capillary beds), the flow will be greatly affected (eg, IVC compression). Arterial afterload, unless extreme, has only a small effect on LV output in preload-dependent hearts with normal LV systolic function (but does have a larger effect in afterload-dependent hearts with decreased LV systolic function). The high CO and low systemic vascular resistance (SVR) that is seen in vasodilatory shock are usually only present after resuscitation when the Pms have been raised with volume and vasopressors. It is only then that SVR falls as the increased flow opens up arterioles. Ultrasound, when just looking at the heart, diagnoses low Pms by showing small volume, hyperdynamic ventricles. It cannot tell you the cause of the low Pms without considering other factors.

In shock, a low CO and SV is detrimental regardless of the cause and must trigger a workup of the mechanism and guide treatment. The key value of echo is that in low CO and SV, it can differentiate Pms responsiveness (PmsR), or PR, from cardiac dysfunction (with some exceptions; see later discussion). The only reason to give fluid in shock is to increase CO and SV. Critical decrease in stressed volume reduces the difference between Pms and RAP and limits SV. A fluid bolus or vasopressor can increase Pms relative to RAP and increase VR and SV (if the ventricles are Pms [or preload] responsive). The authors use the PmsR term, which is considered to be equivalent to the PR term.

Static parameters of cardiac preload (either pressure methods, eg, RAP, PA pressures, E/e' to estimate left atrial pressure (LAP)/left ventricular end-diastolic pressure (LVEDP), or volumetric methods, eg, IVC size, LVEDV) are useful at extremes only. Volumetric methods are more sensitive for low-volume and pressure methods for high-volume scenarios.[42] Dynamic PmsR techniques will inform which patients will respond by increasing their CO. They allow individualized therapy (as opposed to fixed restrictive or fixed liberal fluid strategy). Dynamic tests measure the observed effect of spontaneous or induced cardiac preload changes on CO (or its surrogates, eg, LVOT VTI). Induced changes are achieved by mobilizing endogenous fluid, mimicking fluid challenge (passive leg raising [PLR]), or (using the heart-lung interactions) by variation in cardiac preload induced by mechanical ventilation (variability of LVOT VTI or Vmax). PmsR (PR) tests are listed in **Table 4**.

Several rules apply. First, a PmsR (PR) test is not indicated in very low preload scenarios (hemorrhage, severe hypovolemia) if ample information is available (eg, gross 2D signs of hypovolemia), and the test would delay the treatment. Second, a PmsR (PR) test is indicated only if tissue hypoperfusion is present (fluids or vasopressors are indicated), and the risks of fluids are less than the benefits (fluids would be tolerated). The PmsR (PR) state is physiologic and does not need correcting if there is no hypoperfusion. Third, vasopressors will also increase Pms and VR, for example, in sepsis,[43,44] and should be used instead if the risk of fluid exceeds the benefit (high cardiac filling pressures, end-organ congestion). Fourth, the same PmsR (PR) test can be used during active fluid removal.[45] Fifth, the PmsR (PR) result should be viewed as a continuous variable (the closer to the diagnostic threshold, the less certain about preload response one should be).[46]

Technical limitations exist. Some PmsR (PR) methods perform better than others. ΔSVC and ΔVmax 10% are superior to ΔIVC, compared with PLR as the standard.[47] PLR can be difficult with compression stockings, intraabdominal hypertension, and surgery patients and is contraindicated in intracranial hypertension. Techniques using LVOT VTI variability require mechanical ventilation (see **Table 4**) with tidal volumes greater than 8 mL/kg, passive breathing, and sinus rhythm. RV failure will make PmsR invalid, as significant pulse variations are less likely to predict fluid responsiveness in patients with RV dilatation (variability is from cyclic afterloading of failing [dilated] RV from positive pressure mechanical ventilation rather than reduction of RV preload)[48] (see **Fig. 5**). A spontaneously breathing tamponade patient will have pulsus paradoxus, producing LVOT VTI variability; in mechanically ventilated patients, the variability may be lost. End-expiratory occlusion test effect is small and fleeting and so must be combined with an end-inspiratory test for total ΔLVOT VTI greater than 13%, to get above the diagnostic threshold of echo (Δ>10%).[46] Mini-bolus technique (100–150 mL of colloid or crystalloid greater than 60–120 s) threshold (ΔLVOT VTI 10%) is close to diagnostic precision of echo (least significant change = 10%), so echo may not be precise enough to detect small changes induced by mini-bolus,[49] and increased threshold 15% is recommended.[46] Finally, the authors caution against mandating that every single last drop of preload deficit must be repleted instead of, for example, combining fluid therapy with vasopressor titration, in light of results from large trials where the effect of fluid-liberal therapy was either neutral[50,51] or harmful.[52]

Catastrophic Valve Failure

Severe valvular disease can also cause or contribute to shock. Acute pathologies such as papillary muscle rupture and aortic dissection can cause severe regurgitation, requiring urgent surgical management. Chronic stenosis and regurgitation can have significant effects on the way the heart handles different loading conditions. Infective endocarditis can cause valve incompetence and heart failure.

A major problem with a valve is usually already recognized during the salvage phase. Some valve problems develop secondarily (“functional” regurgitation, eg, MR in cardiomyopathy including sepsis, TR in RV dilation secondary to acute cor pulmonale). More experienced ICU clinicians can use semiquantitative methods during the optimization phase[53] (see **Table 4**). The severity criteria for chronic valve pathologies may not necessarily apply in acute valve failure. However, the authors recommend deferring detailed native or prosthetic valve examination (including quantitation) to consultants or most experienced ICU clinicians because of the potential major treatment decision involved.

Systolic anterior motion of the MV is an important cause of shock in critically ill patients. Basal septal hypertrophy, common in elderly patients, increases the velocity of blood, as it transits the LVOT. A hyperdynamic state, as seen when there is low Pms and sympathetic activation, exacerbates this increased velocity and can suck the anterior MV leaflet into the LVOT, obstructing flow. The resulting effects of the reduced CO can easily be mistaken for failure from reduced contractility. Treatment with inotropes, diuresis, and vasodilators in this case could be catastrophic as immediate treatment should instead consist of volume loading, vasopressors (to increase Pms and VR to fill the heart), and β-blockers (to reduce heart rate and contractility).

Aortic dissection

Aortic dissection can cause shock from aortic rupture and blood loss, tamponade from bleeding into the pericardial space, and severe AR. Although a rare cause of shock, it can be quickly ruled in (or ruled out if images are good) with ultrasound. The whole aorta from the aortic valve down to its bifurcation can be imaged with TTE. Characteristic features include aortic dilatation, a visible intimal dissection flap, pericardial effusion, AR, and turbulent flow.

Left Ventricular Systolic Function

Usually, the heart has reserve compliance—it maintains a low enough volume in diastole to be able to passively fill at a rate depending on the Pms. When failing, it has no reserve, and output becomes dependent on rate and contractility rather than the Pms. Blood volume shifts to the point of the circuit behind the failing ventricle with increased venous pressures (in the lungs in the case of LV failure or the systemic veins in the case of RV failure).

EF is a complex variable depending mainly on LV contractility and LV afterload.[2] It must be interpreted in the context of loading conditions and inotropic support. It is valid as a global evaluation for cardiac (dys)function but not informative on ventricular performance or hemodynamics.[40] In myocardial infarction (MI) and cardiogenic shock, LVEF can be variable.[39]

Mitral annular plane systolic excursion (MAPSE) measures longitudinal movement of myocardium and correlates with EF. Tissue Doppler S′ correlates with dP/dT and is independent of loading conditions. Other linear methods are not typically used because of potential confounding (regional wall motion abnormality, bundle branch blocks, RV dysfunction, beam alignment). Measuring SV directly circumvents shortcomings of LVEF.[54] LV dilation indicates chronicity. Thus, LV dilation points to cardiomyopathy, whereas normal LV size can be seen with acute insults (acute MI, myocarditis, intoxications). Takotsubo, or stress, cardiomyopathy is important to recognize in critical care. It is a nonischamic cardiomyopathy typically causing apical, symmetric regional wall motion abnormalities of the left ventricle, particularly the apex. Its immediate treatment requires discontinuation of B agonists—seemingly counterintuitive when faced with a patient with left ventricular failure. It is difficult to distinguish takotsubo from ischemia without coronary angiography. LVOT obstruction can occur during sepsis.[55] A repeat evaluation of a patient with septic shock whose afterload was increased with vasopressors may reveal an underlying LV systolic dysfunction that was previously hidden by a sepsis-induced decrease in LV afterload.[56] Absent RWMA is usually (but not always) seen with cardiomyopathies, whereas present RWMA is usually seen with acute MI or takotsubo cardiomyopathy. Mechanical complications of acute MI (ventricular septal rupture, free wall rupture and pseudoaneurysms, papillary muscle rupture) should be also considered in differential diagnosis. In

sepsis, LV can be normal or dilated; there can be LV systolic function (global or regional), LV diastolic dysfunction,[57] and RV dysfunction[58] (see **Table 4**).

Left Ventricular Diastolic Function

Impedance to VR can affect VR and CO. Even if CO is maintained, high venous pressures behind the impedance will result. A heart that is unable to relax normally may have a significantly compromised output, especially with acute alterations in loading conditions. Even if CO is maintained, high venous pressures behind the impedance will result. The presence and causes of high impedance are identifiable with ultrasound and have a direct bearing on management. For example, most causes of raised diastolic pressure will respond poorly to fluid administration, resulting in pulmonary edema or systemic venous congestion. Pulmonary artery pressure, or at least the probability of there being raised pressure, can be estimated with various echo parameters.

E/e' as an estimation of LV filling pressures was originally described in cardiology literature[59] and then in mechanically ventilated critically ill patients.[60] It provides only range, not the specific value of pressure. Low E/e' is a good predictor of low filling pressure, and high E/e' is a good predictor of high filling pressure, but 8 to 15 cannot reliably predict LV filling pressure. If indeterminate, many use LA size as tiebreaker, but normal LA size should not exclude LA hypertension especially in acute illness.[61] PEEP can decrease e'.[62] Sometimes, patients with chronically high LAP may not have pulmonary edema. In contrast, patients with relatively normal LAP may have pulmonary edema,[63] making the interpretation of E/e' more complex when integrating echo with findings from lung ultrasound. E/e' does not predict *PR* (**Table 5**). Specifics of echo findings in pericardial constriction are beyond scope of this review.

Right Ventricular Function

In acute cor pulmonale, there is a de novo increase of RV afterload that will affect the RV function. The high pressure and dilatation of the RV flatten the ventricular septum, giving the LV a characteristic D-shaped LV in short-axis views on ultrasound. As the heart is constrained within the pericardium, the LV can only take up whatever space is left by the right heart. Its filling and output are, therefore, impaired. High venous pressures and impaired RV and LV function all contribute to reduced organ perfusion and shock. The degree of echocardiographic impairment can range from RV stress (RV/LV ratio>0.6, RV base diameter >42 mm, D-shaped IVS, septal dyskinesia, McConnell's sign, 60/60 sign, PAT<100 msec) to RV dysfunction (TAPSE <17 mm, S' <9.5, RV fractional area change<35%) to shock (LVOT VTI <14, collapsibility index [CI] <2.2).[64] RV size and RVOT VTI can be used to assess the effects of treatments (volume loading, positioning, pulmonary vasodilators, and mechanical ventilation settings). RV dilatation can be estimated with RVEDA/LVEDA ratio (severe: >1.0).[65,66] RV dilation with dysfunction can be preexisting or acute from PE (involving more than 50% of vascular bed) or acute cor pulmonale related to ARDS and mechanical ventilation[67,68] or sepsis[57,69,70] and COVID-19.[71] RV volume overload results in IVS flattening in diastole, whereas RV pressure overload results in IVS flattening in systole.[72] Demonstrating a thrombus on echo is the only definitive test to prove PE, whereas all other tests can only be suggestive.[73–75] In a shocked patient, normal RV essentially rules out PE as the cause of shock. RV infarct can also result in RV dilation and dysfunction but without high RV afterload and may be associated with LV inferior wall hypokinesia. RV aneurysm can be a clue to arrhythmogenic RV cardiomyopathy, and displacement of the TV leaflet can be a clue to Ebstein anomaly. RV function is measured with TAPSE (like MAPSE, it measures longitudinal movement). Pulmonary artery pressure can be estimated with pulmonary artery acceleration time if there is

Table 5
Preload responsiveness, LV filling pressure, lung and venous congestion in different stages during shock

Ultrasound Parameter	Overt Hypovolemia	Hypovolemia, Preload Responsiveness State	Normovolemia, Fluid Unresponsive State	Hypervolemia
Preload Responsiveness				
Spontaneous breathing				
IVC 2D appearance Collapsibility index (CI)	Small IVC Complete inspiratory collapse (CI test not necessary)	CI ≥48%	CI <48%	Dilated IVC >2.1 cm CI <<< 48% to no inspiratory collapse
Mechanical ventilation				
IVC 2D appearance Distensibility index (DI)	Small IVC <1 cm Significant inspiratory distension (DI test not necessary)	DI≥12%–18%	DI <12%–18%	Dilated IVC (unclear threshold in cm) DI <12%–18% to no inspiratory distension
SVC (TEE) 2D appearance Collapsibility index (CI)	Triangular vessel Significant inspiratory collapse (CI test not necessary)	CI ≥36%	CI <36%	Dilated vessel CISVC <<<36% to no inspiratory collapse
ΔLVOT VTI or ΔAoVmax respiratory variability	Significant variability (test not necessary)	ΔLVOT VTI>18% ΔAoVmax>12%	ΔLVOT VTI<18% ΔAoVmax<12%	No variability
Spontaneously breathing or mechanically ventilated				
ΔLVOT VTI during passive leg raising	(test not necessary)	≥12%	<12%	No Δ
LV filling pressure				
LV appearance	Small LVEDA End-systolic LV obliteration	Does not predict	Does not predict	Dilated LV Ballooning LV
LV filling pressure (2D signs)	Small LVEDA End-systolic LV obliteration	Does not predict	Does not predict	Continuous bulging of interatrial septum
Mitral inflow	Test not necessary	Does not predict	Does not predict	E/A>2 (restrictive)

(*continued on next page*)

Table 5
(*continued*)

Ultrasound Parameter	Overt Hypovolemia	Hypovolemia, Preload Responsiveness State	Normovolemia, Fluid Unresponsive State	Hypervolemia
Mitral E/e' (mean) (mitral inflow velocity/ tissue velocity)	Test not necessary	Does not predict	Does not predict	E/e'>14
Lung congestion				
Lung ultrasound pattern A-pattern (bilateral A lines) B-pattern (bilateral B lines)	A lines	Unknown[a]	Unknown[a]	Most commonly B-pattern[b]
Venous congestion				
Hepatic vein			S>D[c]	S<D or S reversal
Portal vein	Test not necessary	Test not necessary	Continuous flow Low pulsatility (PF 30%–49%)[c]	Increased pulsatility (PF>50%) Cyclic reversal
Renal vein	Test not necessary	Test not necessary	Continuous flow, monophasic[c]	Discontinuous flow, S + D Discontinuous flow, D only
Femoral vein	Test not necessary	Test not necessary	Continuous flow Absent or minimal respiratory variation[c]	Increased pulsatility Cyclic reversal

[a] Not studied for this indication.
[b] Some patients with higher PAOP may have A-pattern. B-pattern correlates with lung congestion (increased EVLW), less with intravascular congestion (PAOP).
[c] VeXUS scoring applies when IVC size greater than 2.1 cm.
Bold: essential information; Not bold: further expansion of information.

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no TR.[76] The midsystolic notch is another useful clue for pulmonary hypertension. PASP may not be reliable during RV dysfunction (RV cannot generate high pressure). Fluid management in RV failure is important since hypo and hypervolemia are detrimental. In the presence of RV contractile dysfunction with normal RV afterload (RV infarction), one can aim for a higher preload. On the contrary, during high RV afterload, volume can displace IVS, impair LV filling, and reduce CO (via ventricular interdependence). Reducing RV preload is important in such a situation, and serial echo to guide fluids, or inotropes rather than vasopressors, is useful[77] (see **Table 4**).

Distributive Shock

Distributive shock may present with no apparent abnormality on echo (after correcting hypovolemia). It may also be present and driving the shock state in patients with structural cardiac dysfunction. As discussed earlier, LVOT VTI and PLR can help differentiate between distributive shock (normal or high VTI, transient PmsR present), hypovolemia (low VTI, sustained PmsR present), and cardiogenic or obstructive shock (low VTI, PmsR absent).

STABILIZATION AND DEESCALATION PHASES

Fluid Accumulation

Resuscitation in shock often leads to fluid overload. One definition is that of greater than $\geq$10% body weight fluid accumulation. Rather than all-or-none phenomenon, it should be conceptualized as a continuum, as no specific threshold of fluid balance can capture fluid overload in all individuals. Fluid overload is not synonymous with hypervolemia.[78] It may occur with intravascular hypovolemia, normovolemia, or hypervolemia, it may or may not be associated with interstitial edema, and it may or may not be associated with globally increased permeability. It has been suggested that the term "fluid accumulation syndrome" (as any degree of weight accumulation resulting in a pathologic state of overhydration associated with clinical impact and worse outcomes) should be used.[11] The association between positive fluid balance and mortality is well known.[79,80] In an ideal scenario with carefully titrated fluid resuscitation, fluid overload might be avoided. From large observational studies and clinical experience, fluid overload does occur, however. Surprisingly, the actual incidence of fluid accumulation syndrome in the era of personalized ultrasound-directed resuscitation during early shock phases has not been well studied. Physiologically, hypervolemia leads to increased pressures and edema, increased pulmonary artery pressure leads to RV dysfunction, and increased venous pressure leads to impaired organ perfusion pressure.

Weight change, cumulative fluid balance, or impedance measurements are nonultrasound markers of fluid accumulation.[11] Ultrasound techniques are very useful in detecting end-organ congestion (lungs and venous system). None of the ultrasound techniques (measurements of pressures, volume, PmsR [PR], extravascular lung water, and venous stasis) exactly define volume status and should be used as complementary when combined (see **Table 4**). Details of relevant ultrasound techniques (lung ultrasound, systemic venous congestion) are beyond scope of this work and can be found in the corresponding article.

Fluid Removal Tolerance

When intravascular volume is excessively reduced, SV and blood pressure decrease, negatively affecting end-organ perfusion. Intravascular volume depends on oncotic pressure, blood pressure, and vascular permeability. As intravascular blood volume

is removed, intravascular refill (2–6 mL/kg/h or even higher in healthy patients) maintains a net balance. Too aggressive fluid removal (net ultrafiltration rate > 1.75 mL/kg/h) was associated with worse outcomes,[81] but so was also low-intensity net ultrafiltration.[82] Therefore, fluid removal should not be too aggressive and should be done only in patients who can tolerate it. Tolerance is defined hemodynamically (decrease of systemic pressure, SV) or based on perfusion (increased lactate). Some degree of hypotension requiring vasopressor support is sometimes acceptable if it is not causing tissue hypoperfusion. No single clinical marker alone (fluid balance, central venous pressure, or clinical endpoints like extubation, oxygenation improvement, or chest X ray improvement) can predict tolerance and guide fluid removal. However, PmsR (PR) techniques can predict which patients would not tolerate fluid removal. PR of 9% change of CI (measured during passive leg raising test with transpulmonary thermodilution with pulse contour technique) predicted which patient would develop intradialytic hypotension.[83] The echocardiography-guided PLR PmsR (PR) technique uses the same principle, although it would need to adjust the diagnostic threshold (as 9% in the original study is below the least change detectable by echo).

LIMITATIONS AND CAVEATS OF ECHOCARDIOGRAPHY IN SHOCK

Echocardiography is not a harmless tool. Wrong conclusions can lead to wrong treatments, posing a potential patient risk.[84] ICU clinicians should be aware of several possible diagnostic errors. One problem is the availability bias.[85] Examples include administering tissue plasminogen activator for RV failure from ARDS (thinking “PE”) while the actual diagnosis is ARDS-induced acute cor pulmonale; starting an inotrope in a patient with decreased LV EF (thinking “cardiogenic shock”) who has underlying cardiomyopathy, a normal SV by LVOT VTI, normal or even high Scvo2, and has distributive shock from sepsis; or inserting a chest tube for absent lung sliding in an unstable patient while the actual problem is unilateral mainstem intubation. Several other human factor sources of errors exist. These are not being systematic during examinations, not considering all available relevant clinical information, ignoring changes in patient status, not doing repeat examinations, not applying techniques correctly, or ignoring limitations of the techniques. Echocardiography also has inherent limitations independent of the operator (quality of views available, technique limitations). Sometimes, it is not possible to pinpoint shock diagnosis to a single mechanism. Although a unique and powerful tool, echocardiography is not a standalone tool in shock management. It is not a continuous hemodynamic monitor.[86] It is complementary to other techniques, including physical examination, hemodynamic monitoring devices, and markers of tissue perfusion.

ULTRASOUND IN SHOCK AND PATIENT OUTCOMES

Because echocardiography is considered essential for shock management, ICU clinicians should be aware of current knowledge about its impact on important outcomes such as morbidity and mortality. The authors briefly review evidence emphasizing randomized prospective studies and patient-centered outcomes, such as the impact on mortality and morbidity. This evidence is still evolving. The authors will disregard a large body of literature showing the well-known positive effect of the ultrasound on intermediate outcomes such as diagnostic accuracy, feasibility, and change of management.

The impact of ultrasound on shock outcomes was studied in the most common form of shock (septic) and undifferentiated shock (a large portion of which were patients with septic and cardiogenic shock).

In septic shock, retrospective or prospective observational studies showed association with improved outcomes[87–92] versus no effect on outcomes[93,94] versus worsened outcomes.[95] In randomized trials, the most consistent finding is that fewer fluids are given, and there is a trend toward lower mortality in ultrasound arms.[96–105] Recent meta-analyses on dynamic tests of PR either showed an association with decreased mortality, length of stay, and duration of mechanical ventilation[106] or did not show improved outcomes.[107,108] Surviving sepsis guidelines recommend resuscitation being guided by dynamic over static measures.[109] A focused Society of Critical Care Medicine (SCCM) updated on echocardiography in septic shock has been recently published.[110]

In the undifferentiated shock (of which septic shock is a significant portion), retrospective or prospective observational studies showed either no effect[111–113] or worsened outcomes.[114] In randomized trials, there was no effect on outcomes.[18,115]

These results should be interpreted in the context of the accumulating knowledge over the past several decades on hemodynamic interventions in sepsis. It is difficult to prove an improvement in hard outcomes in such a heterogeneous population of patients where other protocolized hemodynamic interventions in large trials consistently showed no significant impact compared with usual care[50,116] and where fluid restrictive or fluid liberal management resulted in the same outcomes.[51] The techniques and treatments are often not standardized. Patients may enter trials during different phases of shock, which matters as echocardiography and ultrasound goals change over time. Although the evidence on management impact during the deresuscitation phase is developing,[117–119] the role of ultrasound in management has not been rigorously studied. However, in the critical care echocardiography community, there is consensus that, especially in experienced hands, echocardiography is a powerful tool that allows a unique view into the mechanism of shock, optimization of therapy in the later phase of shock, and assessment of organ congestion in later phases. Compared with a one-size-fits-all approach, it offers a personalized and presumably better approach to treating patients in shock.

CLINICS CARE POINTS

- Clinicians proficient in critical care echocardiography should be able to acquire diagnostic-quality images, identify the most likely diagnosis while considering relevant differentials, and formulate a treatment plan—often implemented immediately—all at the bedside.
- During the resuscitation phase of shock, echocardiography should be focused, goal-directed, and qualitative, relying on visual pattern recognition. In the optimization phase, a semi-quantitative or quantitative approach is used, grounded in physiological principles.
- Assessment of preload responsiveness can guide both fluid administration and vasopressor therapy by evaluating changes in mean systemic pressure and venous return.
- Estimation of stroke volume via LVOT VTI is now considered a fundamental echocardiographic skill. While advanced quantitative techniques may be applied in any phase depending on clinician expertise, they are typically reserved for later phases of shock management.
- Many patients can be stabilized using only basic echocardiographic techniques; however, to fully harness the full potential of critical care echocardiography, clinicians should become proficient in advanced methods.

DISCLOSURE

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